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Blast injury in enclosed spaces

All doctors should know the basic management of patients injured by explosive blast

The bomb attacks on the transport network in London on 7 July 2005 have illustrated the lethality of explosions in confined urban spaces. Such indiscriminate attacks could occur again in the near future. The management of casualties injured by blasts is mainly the preserve of the military doctor, but the bombing of a bus outside BMA House graphically illustrates that any doctor may be called on to manage patients injured in explosions.

While all casualties injured in explosions should be managed initially according to the advanced trauma life support (ATLS) guidelines, starting with airway, breathing, and circulation,[1] general doctors should also be aware of the specific features of blast injury. Detailed advice and clinical guidance for the non-specialist on managing casualties injured by blasts is freely available online from the Centers for Disease Control and Prevention in the United States.[2]

Injuries caused by explosive blast were classified by Zuckerman during the second world war according to the physical effects on the body caused by the released energy.[3] Primary injuries result from the interaction of the blast shock wave with the body and affect the areas where air and tissue meet, including the ear, lung, and the gut. Secondary injuries result from the collision of energised fragments with the body. Tertiary injuries result from displacement of the whole body or body parts by the blast energy, and include traumatic amputations. In addition, tissues are burned by the hot gases from detonation and from inhalation of smoke and debris in aerosol form.

Immediate clinical manifestations of acute lung injury include pneumothorax, pulmonary oedema, and air embolism. Delayed effects over the next 24-48 hours create a picture similar to acute respiratory distress syndrome, with hypoxia and diffuse infiltrates in the lung, and high mortality.[4] Traumatic amputation of a limb, as a marker of severe multisystem injury, also has high mortality.[5] Tympanic perforation is common in the survivors of blasts at close proximity, but damage to the eardrum without obvious signs of other injuries does not seem, on the basis of one observational study, to be associated with more serious morbidity such as lung damage.[6]

Simple blast waves in an open space create a rapid rise in air pressure usually lasting less than 10 milliseconds. In enclosed environments the reflection of blast waves from walls and other surfaces creates complex waves of longer duration. This allows greater transfer of energy to the body, increasing the risk of primary blast injuries such as tympanic perforation and blast lung[7] and increasing displacement of the body wall, which may cause a shearing effect on larger organs, especially abdominal viscera.[8]

In two explosions in the open air in Israel, mortality among casualties who required treatment in hospital was 8% (15 deaths among 204 casualties), whereas after two blasts in the enclosed space of a bus 49% of patients (46 deaths out of 93) eventually died.[9] In addition, the severity of injuries among the survivors was higher in the bus bombing as graded by the injury severity score (mean score 4 after a blast in the open air compared with 18 after a bus bombing, with a greater proportion of the casualties from the bus having primary blast injuries: 34% in the open air compared with 78% on the bus). Similar observations were made among casualties when the Irish Republican Army (IRA) bombed pubs in the United Kingdom in the 1970s.[10]

The bombings on 7 July in London caused a large number of casualties (around 700), but most injured people were discharged from hospital soon after assessment and treatment. Many were injured by non-penetrating fragments, which can be managed without surgery.[11] In an uncontrolled incident, vast numbers of "walking wounded" can lead to a "reverse triage effect" where patients with minor injuries present to hospitals before the serious casualties arrive, swamping emergency services to the detriment of the severely wounded.[2] That this phenomenon did not occur on 7 July testifies to the outstanding integrated response by the pre-hospital services in controlling actions at the scenes of the incidents and in triaging patients appropriately.

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Preventing blindness from glaucoma

Better screening with existing tests should be the priority

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The detection and management of primary open angle glaucoma is a major healthcare issue. It is the second largest cause of blindness in the world and affects some 66.8 million people, leaving 6.7 million with bilateral blindness.[1] In the United Kingdom, the ageing of the population means that the number of cases is expected to increase by 30% in the next 20 years.[2]

In primary open angle glaucoma, the retinal ganglion cells—the nerves that carry the visual stimulus from the retina to the brain—undergo apoptosis after insult at the head of the optic nerve. The progressive loss of ganglion cells leads to characteristic structural changes at the head of the optic nerve and functional loss to the visual field. Glaucoma is often, but not necessarily, associated with raised intraocular pressure. A paper in this week's *BMJ* shows that, in general, treatment to reduce intraocular pressure leads to delayed progression of visual field loss in patients with manifest open angle glaucoma.[3] More research is needed in the subgroup of patients without increased intraocular pressure, to determine which patients with normal tension glaucoma will benefit most, since this meta-analysis was unable to show a consistent benefit in these patients.[3] In 1982 Grant and Burke wrote a paper intriguingly titled "Why do some people go blind from glaucoma?"[4] From a sample based in the United States, they found that some 30% of people who go blind from this disease are blind, in both eyes, at presentation. Most of the blind patients were aware of their decreasing vision for months, or even years, before they sought medical advice. Blindness was defined as an acuity of less than 20/200 (< 6/60 metric Snellen) in the better eye, or a residual visual field of less than 10 degrees. In a more recent report by Sinclair,[5] who investigated registrations for blindness due to glaucoma in Fife between 1990 and 1999, a considerable number of patients were found to have moderate to advanced visual field loss at their first appointment, with 23% being eligible for registration as blind.

We recently reviewed all referrals for glaucoma and registrations for blindness or partial sight at Manchester Royal Eye Hospital during 2003. We found that 28% of patients with glaucoma were registered blind within three years of first presentation and that relatively few of those with blindness or partial sight were referred initially by optometrists: 42% compared with 90% nationally for all people with suspected glaucoma (unpublished data).This indicates that there may be barriers to access, such as the perceived costs associated with getting an eye examination. Laidlaw has already shown that the imposition of fees for sight tests had a negative effect on the number of referrals to Bristol Eye Hospital for glaucoma.[6] New technologies, such as optic nerve and nerve fibre layer imaging devices, are promoted on the basis of being able to detect glaucoma before the patient has a reproducible visual field defect (because, unsurprisingly, those with more rapidly progressing disease leading to blindness are more likely to present with marked visual field loss[7 8]). But new technologies are not required to detect the extensive visual field loss that many of those who progress to blindness have at first presentation: the tests we already have are capable enough if used appropriately.

A series of epidemiological studies has shown that the more widespread use of existing technologies will improve early detection. In the north London trial,[9] 75% of cases with "definite" glaucoma were new cases, and these were detected with a simple combination of tests—suprathreshold perimetry, tonometry, and slit lamp examination of the anterior eye and optic nerve head—that are readily available at most optometric practices.

The problem is not lack of suitably sensitive technologies but the infrequent use of existing technologies. Breaking down barriers to access, targeted screening, and a campaign to inform patients about the importance of regular eye examinations might have much more effect on the number of patients going blind from this disease than the current concentration of effort into the development of more sensitive technologies.

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